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EFFECTS OF ETHANOL AND ADAPTATION UPON EYEBLINK CONDITIONING

by

Gordon Neville Hobson

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Effects of Ethanol and Adaptation upon Eyeblink Conditioning", submitted by Gordon Neville Hobson in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

ABSTRACT

The aim of this study was to investigate the effects of ethanol and adaptation upon eyeblink conditioning, thereby acquiring data with which to evaluate hypotheses derived from the theories of both Eysenck and Spence.

The specific hypotheses were:

1) If, as a consequence of repeated trials using the unconditioned stimulus alone, Spence's response mechanism adapts and causes a decrease in drive level; and if further, the same process reduces excitatory potential and increases inhibitory potentials as Eysenck defines these constructs, it is predicted that, regardless of ethanol dosage, the acquisition and resistance to extinction of the conditioned eyeblink response will be inversely related to the number of adaptation trials.

2) If the depressant drug ethanol increases cortical inhibition, and decreases cortical excitation, as Eysenck maintains, and if further, this same drug produces a reduction in emotional responsiveness, which mechanism, according to Spence, directly influences drive level, it is predicted that, regardless of the number of adaptation trials involved, acquisition and resistance to extinction of the conditioned eyeblink response will in both cases be inversely related to ethanol dosage.

3) Since ethanol is presumed to be a central nervous system drug which depresses performance, if adaptation is a similar inhibitory process, it is predicted that acquisition and resistance to extinction of the conditioned eyeblink response will be affected similarly by both variables.

To test these hypotheses, 135 Ss were randomly assigned to 3 ethanol dosages and to 3 levels of adaptation, leading to 9 treatment conditions with 15 Ss in each sub-group.

Analysis of variance of the results confirmed all three hypotheses at the 0.01 level of significance as they related to response acquisition. The hypotheses related to extinction were not confirmed.

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Gordon Neville Hobson.

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CHAPTER 1

Introduction

In the psychology of learning, a major area of research has been concerned with variables that have an effect upon the strengthening and weakening of response tendencies. Experiments with human subjects have provided evidence that personality characteristics may be an important variable in the above process (Child and Waterhouse, 1953; Lazurus and Ericksen, 1952; Mandler and Sarason, 1952). Operating specifically within a learning theory framework, Eysenck (1957, 1963), Franks (1956, 1957, 1963), Spence (1954, 1958, 1960) and Taylor (1951), have attempted to demonstrate the relationship between personality variables and eyeblink conditioning.

The purpose of the present undertaking was to provide data to evaluate hypotheses derived from Eysenck's personality theory and from Spence's theory of emotionally based drive, as well as to permit investigation of the relationship between these two theories. To this end, the effects of ethanol and the effects of adaptation upon the acquisition of the conditioned eyeblink response were studied. Additionally, this experiment was designed to facilitate an understanding of the action of the independent variables, per se, upon the conditioning process.

The Independent Variables

Essentially this was an empirical study of drug and non-drug produced inhibition upon eyeblink conditioning. Three dosages of ethanol, and three experimentally manipulated levels of adaptation, were selected as the independent variables.

Ethanol. This drug was chosen for several reasons: a review of the appropriate literature disclosed that few reliable studies of the consequences of this variable upon human behavior have been reported, and practically none involving human conditioning. According to Carpenter (1961) and Drew (1963), little progress has been made in the past 25 years toward developing a knowledge of the effect of alcohol upon the learning process.

Ethanol, as a known depressant drug having inhibitory effects upon the central nervous system, can also be directly utilized to test hypotheses derived from Eysenck's theory, in which individual differences in personality are attributed to constitutional differences in a cortical excitatory-inhibitory balance. According to Eysenck (1957), a depressant drug will predictably retard acquisition of the conditioned eyeblink response through its effects upon the hypothesized cortical mechanism. The results of studies using depressant drugs other than ethanol have generally supported Eysenck's claims.

Since the depressant effects of ethanol are generally accepted to be anxiety reducing the action of this drug upon conditioning has relevance for Spence's hypothetical emotional response mechanism, operationally defined in terms of manifest anxiety. It would be expected that if the level of emotional responsiveness were to be reduced by ethanol, a corresponding reduction in drive would be reflected in conditioning performance.

Adaptation. The process of adaptation was seen to have equal applicability to the present undertaking. From a commonsense point of view, adaptation would seem to have an inhibiting effect upon

behaviour. It is a frequent observation, for instance, that repetition of a mildly noxious stimulus appears to inhibit its own effectiveness, as indicated by the diminishing response made to it. On the assumption that it is an inhibitory mechanism, the effect of experimentally controlled degrees of adaptation upon eyeblink conditioning has consequence for both theories being investigated. In the case of Eysenck, an increase in inhibition should predictably lead to "extraverted behaviour", defined in terms of eyeblink response decrement. For Spence (1958), such a procedure is a direct test of his adaptation theory. It would be expected that adaptation of emotional responsiveness to the unconditioned stimulus would lead to a reduction in the drive level, and therefore in conditioning performance.

Finally, this experiment was designed to permit study of the phenomenon of adaptation for its own sake. It was hoped to gain knowledge about the relationship between the action of a known depressant drug and the adaptive mechanism; similarly, about the way in which adaptation affects response decrement in conditioning studies which employ noxious stimuli. In regard to the latter, as Martin (1964) points out, psychologists concerned with the formulation of general behaviour schemata have been preoccupied with the positive aspects of reaction to the exclusion of equally important reciprocal or negative reactions. A review of the appropriate literature, in which can be found very few investigations of adaptation, supports this assertion.

Organization of the Dissertation

This research potentially relates to such disciplines as psychology, pharmacology, neurophysiology, biochemistry, and the all-embracing field of alcoholism research. Since the specialized literature in these areas is beyond the scope of the present undertaking, the organization of this dissertation will take the following form: in chapter one, a short history of the concept of inhibition from a psychologist's point of view is followed by a detailed discussion of Clark Hull's behaviour theory. This discussion deliberately emphasizes the problems related to inhibition, a theme developed further in a review of Eysenck's theory. The chapter concludes with some theoretical considerations of the two independent variables, ethanol and adaptation, with special reference to Spence's theory.

Chapter two presents the relevant experimental literature in the following manner: the phenomenon of adaptation; studies of the general effects of drugs (ethanol and others) upon conditioning; studies in which depressant drugs other than ethanol were used to test Eysenck's theory; and finally, a critical analysis of the only reported study in which ethanol was used to test this same theory. Chapters three, four, five and six, based upon the above considerations, present respectively the description of the present experiment, the results, and the discussion.

CHAPTER 2

Theoretical Considerations

The hypothetical construct of inhibition is the common denominator of the various facets of this investigation. In both classical and experimental conditioning experiments, for example, inhibition has long been regarded as a major variable having a marked effect upon performance. In the case of ethanol, as a depressant drug its action is believed to be of an inhibitory nature. Finally, and as pointed out earlier, the process of adaptation appears also to have an inhibiting effect upon behaviour.

Since the concept of inhibition is used in different ways by different investigators, it is necessary to state that this study was concerned only with Hullian inhibition theory, and its interpretation or modification by Eysenck and Spence. In this chapter, the historical origin of inhibition as it has involved psychology will be noted briefly before its role in modern learning theory is commented upon.

Historical origin of inhibition

> When physiologists have solved the problem of inhibition, they will be in a position to consider that of volition. (p. 406).

This quotation, from Animal Life and Intelligence, by Lloyd Morgan, (1891) was made at the beginning of a decade when psychologists were more interested in the theoretical significance of physiological inhibition than at any time before or since. The apparent unity between the two disciplines was shortlived. In the view of at least one historian, psychologically based learning theorists were

created for the sole purpose of repudiating the physiological roots of the inhibitory process. According to critics such as Diamond, Balvin and Diamond (1963) and Wolman (1960), the price paid for such neglect is reflected in what is claimed to be the contemporary "sterile conditioned reflex approach to behaviour analysis." This notwithstanding, it is paradoxical that the core of modern learning theory is founded upon the techniques of the physiologist Pavlov, who apparently thought little of psychologists and psychology. The importance of conditioning and extinction to learning theory makes it appropriate to summarize the Russian's contribution.

What goes on in the central nervous system, according to Pavlov, can be described as a dynamic balance of energy involving such processes as stimulation, inhibition, irradiation and concentration of energy. Interpreted in terms of the conditioning process, reinforcement is assumed to increase the power of conduction of neural connections between the conditioned stimulus (CS) and the response. This increase in so-called conductivity is known as excitation. Conversely, a decrease in responsiveness (such as is evident in the extinction process) is attributable to the action of inhibition. Bereft of its physiological content, such terminology is common to the description of contemporary learning and extinction curves. For example, a growth in an acquisition curve is often referred to as a growth in excitatory potential, whereas a decrement in response is held to represent a growth in cortical inhibitory potential.

While several types of inhibition were postulated by Pavlov, that which he called internal inhibition has had the greatest impact upon learning theory. This phenomenon occurs under definite and specified conditions wherein the positive CS, itself, becomes inhibitory, and invokes in the cortex a process of inhibition instead of excitation. In Hull's system reactive inhibition (I_R) appears to be closely related to Pavlov's internal inhibition, and a similar concept is expressed by Eysenck (1957) in temporal inhibition (D-).

In spite of the detailed nature of Pavlovian theory, several crucial criticisms have undoubtedly helped to curtail its influence as a physiological theory upon the discipline of psychology. The concepts of inhibition and excitation, for example, far from being derived from direct measures of cortical functions, are inferred from overt movements or quantities of secreted saliva. Additionally, these physiological concepts are based upon the premise that conditioning is exclusively a cortical function. As Hilgard and Marquis (1961) and Osgood (1953) state, many experiments have clearly shown that conditioned responses can be established in invertebrates lacking typically cerebral structures. Also, incontrovertible demonstrations (Bromley, 1948; Girden, Mettler, Finch and Culler, 1936) have shown that conditioning takes place in higher animals rendered decorticate by surgery.

Although Pavlov's neurological theory was not accepted by the psychological behaviourists, the conditioned reflex (CR), immortalized in North America by Watson (1913) assured the Russian scientist a place

in psychological history. In 1915, in his presidential address to the American Psychological Society, Watson put all his efforts into extolling the place of the conditioned reflex in psychology. This event, in Hilgard's (1956) opinion, began a non-physiological interpretation of Pavlov which endures to the present day.

The Contribution of Clark Hull

While obviously not unaware of Pavlov's contribution, it was apparently the appearance of the first translation of the Russian's work by Anrep (1927) that captivated Clark Hull. In 1929, Hull stated the theme that was to dominate all his future efforts when he described Pavlov's conditioned reflex as:

> . . . an automatic trial and error mechanism which mediates, blindly but beautifully, the adjustment of the organism to a complex environment. (p. 498)

While hopeful of molecular interpretation, investigation of behavioural phenomena, Hull thought, had to be conducted at the molar level. In 1935 he proposed that the majority of Pavlovian experiments might be considered as a special case under Thorndike's law of effect. Two years later, he was to publish a formal dissertation of Pavlovian conditioning based on reinforcement principles. It is quite frequently forgotten, however, that Hull, often referred to as a "peripheralist", was ever aware of the importance of physiological theory. In 1943, while of the opinion that current knowledge of physiological processes was inadequate for the development of a molecular theory of behaviour, he had this to say:

> It is conceivable that the elaboration of a systematic science of behaviour at the molar level may aid in the development of an adequate neurophysiology, and thus lead to . . . a truly molecular theory of behaviour firmly based on physiology. (p. 20)

As is well documented, during the past 30 years, Hull's systematic behaviour theory has generated a vast amount of research concerned with the learning processes. Much of this research has been focussed upon inhibition mechanisms and their effect upon performance. As a topic central to the present study, it will be discussed as it first involves Hull, then Spence, Eysenck and others.

Most of Hull's behavioural laws are derived either from classical or instrumental conditioning experiments. Although the system has undergone constant change by Hull (1943, 1951, 1952), and by such as Jones (1958) and Spence (1958), the needs of the present enquiry can be met by reviewing the main postulates.

The central learning concept in Hull's theory is habit strength ($_{S}H_{R}$), the nature of which is determined solely by the number of reinforcements. In turn, $_{S}H_{R}$ is multiplicatively associated with those remaining variables in the system held to be directly related to reaction potential ($_{S}E_{R}$). Thus, for any given level of $_{S}H_{R}$, the magnitude of $_{S}E_{R}$ depends upon four other variables--a relationship illustrated in the basic behaviour formula:

$$_{S}\bar{E}_{R} = {}_{S}H_{R} \times D \times V \times K \times J$$

Of these four variables, D, which represents primary drive, is the most important. Hull conceived this to be a general rather than

specific behaviour energizer. At any given time ${}_SH_R$ may be sensitized into ${}_SE_R$ by all the primary drives active within the organism: ${}_SE_R = {}_SH_R \times D$. As will be shown later, this same concept is amplified by Spence and given a basis in emotional responsiveness. In regard to the remaining variables, V represents stimulus intensity, K the magnitude of the reinforcement, and J an inverse function of the delay in reinforcement.

While each of these four variables is related positively to ${}_SE_R$, opposing inhibitions may be encountered which tend to prevent response evocation. This condition is shown in the lengthened behaviour formula:

$$ {}_S\bar{E}_R = {}_SH_R \times D \times V \times K \times J - I_R - {}_SI_R $$

the added symbols representing primary negative drive and conditioned inhibition respectively, Hull's two basic inhibition concepts.

The need for these latter intervening variables stemmed from the observation that as ${}_SH_R$ increased with the number of reinforcements, it decreased with the number of non-reinforced trials. Hull (1952, p. 8) stated that, reinforced or not, any reaction evoked from an organism results in an increment of primary negative drive (I_R) which inhibits ${}_SE_R$ according to its magnitude. This concept of reactive inhibition, most frequently thought of as a work decrement, had its origins in the work of Miller and Dollard (1941), Mowrer and Jones (1943), and Pavlov (1927).

Long term extinction, called by Hull conditioned inhibition (${}_{S}I_{R}$) became a special case of the tendency of all non-reinforced responses to develop a barrier to their own repetition. Repeated trials of the stimulus-response sequence is held to result in a "build-up" of I_R. This, when observed to dissipate with rest, was assumed to be a condition reinforcing to the inactive state resulting in a "conditioned-not-responding". Thus I_R is a negative drive which has a non-permanent effect upon ${}_{S}E_{R}$, whereas ${}_{S}I_{R}$ produces a permanent reduction in ${}_{S}E_{R}$.

A Criticism of Clark Hull

In some ways, this is as far as the development of this particular inhibition theory has gone. Hull's formulations have been criticized and found wanting. Many of these criticisms have their origin in investigations of extinction phenomena. Hull's inhibition theory, for instance, has been found inadequate to explain the extinction of relatively effortless galvanic skin responses, or the resistance to extinction of conditioned fear responses--which usually involve effortful work. Studies conducted with drugs such as curare (Black, 1958) in which extinction takes place without responses being made, are also a typical challenge to Hull's system.

At a more theoretical level, Hilgard (1956), in claiming that Hull's inhibition theory was the least satisfactory part of his system, expressed the opinion that if I_R is claimed to be a negative drive, it should logically subtract from positive drive:

$$ {}_{S}\bar{E}_{R} = \left[\left(D - I_R\right) \times {}_{S}H_{R}\right] - I_R \times {}_{S}I_{R} $$

Iwahara (1957), attacking the lack of internal consistency in Hull's theory, asserted that the relationship between drives and habits should always be multiplicative and never additive, thus:

$$_{S}\bar{E}_{R} = (_{S}H_{R} \times D) - (_{S}H_{R} \times I_{R} \times {}_{S}I_{R})$$

A different approach altogether was taken by Spence (1958), a former student of Hull. Instead of trying to modify Hull's inhibition theory, he argued that inhibition should be related only to non-reinforced trials and should, therefore, be thought of as extinctive inhibition (I_n). Unlike Hull's I_R, and probably not even related, I_n subtracts from $_{S}E_{R}$, and in no way interacts with other intervening variables. Such a proposal, it is noted, may just as easily be interpreted as an abandonment of the original problem rather than a resolution of it.

Jones (1958), in what may be the most radical departure from Hull's original formula, stated that if $_{S}I_{R}$ is simply the habit of not responding due to the reinforcing effect of I_R dissipation, then logically $_{S}I_{R}$ should be deducted from $_{S}H_{R}$:

$$_{S}\bar{E}_{R} = (D - I_{R}) \times (_{S}H_{R} - {}_{S}I_{R})$$

According to Jensen (1961) in his review of the pertinent literature, empirical findings do not support any of the aforementioned or similar suggested modification of Hullian theory. It is thus

apparent that neither Hull's original inhibition variables nor the revisions are adequate to account for the inhibition process. In the present author's view, it would seem that preoccupation with algebraic manipulation of these variables not only forces theorists into indefensible positions, but also stifles creative speculation about what other factors might be involved in inhibition. In regard to the former, since Hull holds that I_R and $_SI_R$ subtract from $_SE_R$--a performance variable--how can the extinction of former habits be explained? The assertions made by both Hilgard and Iwahara--that I_R and $_SI_R$ are multiplicative variables--again defy understanding. How can the result of the interaction of two variables which, by definition, are response inhibiting, result in a positive addition to $_SE_R$?

These theoretical difficulties, further compounded by negative empirical findings, should be sufficient to cause investigators to explore the possibility that variables other than, or in addition to, I_R and $_SI_R$ are needed to explain inhibition. Several facts seem to be clear: I_R does not occur in all cases; frequently, in those cases in which it does occur, I_R dissipation does not lead to $_SI_R$, and this in turn, points to the inadequacy of $_SI_R$ as an explanation for permanent extinction. An inhibition theory must, therefore, account for extinction of responses when none are made, the non-extinction of responses when much "work" is involved, and the permanent change in $_SH_R$ when the $_SI_R$ concept is inapplicable.

To this end, the concern with algebraic manipulation of two inhibition constructs might profitably be forsaken in the interests of

investigation of the relationship to inhibition of such variables as the modality of the response, the simplicity or complexity of the task, species, and, when appropriate, cognitive factors. Many theorists seem to be caught up in the paradox of seeking to present a formula to cover all cases without bothering first to find out what all the cases are. It is the present author's belief, for instance, that all human inhibition phenomenon will ultimately be found to be a combination of cortical and peripheral (localized) processes, to a large measure influenced by cognitive factors.

Eysenck--Inhibition and Personality Theory

The reformulation of the inhibition concept by Jones (1958) was adopted by Eysenck as the basis for a personality theory derived from learning principles. In this theory, positive drive (D) is grouped with negative drive (I_R), and positive habit ($_SH_R$) with negative habit ($_SI_R$), resulting in the formula:

$$_S\bar{E}_R = f(_SH_R + {}_SI_R) \times f(D) + I_R$$

This formula is expanded yet further as a result of Eysenck's expressed aversion to the use of the symbol I_R. This, he considers, has connotations such as peripheralism and dependency on amount of work done, with which he has little sympathy. Consequently, positive drive becomes D+, and I_R, conceptualized as a central nervous system phenomenon having drive properties of a negative character, as D-. The final formula thus takes the following form:

$$_S\bar{E}_R = f(D+ \times {}_SH_R) - f(D+ \times {}_SI_R) + f(D- \times {}_SH_R) - f(D- \times {}_SI_R)$$

Despite the painstaking efforts to put inhibition it its proper place, and in spite of his criticisms of the neo-Hullian peripheral treatment of inhibition, Eysenck fails himself to be consistent in his interpretation of this variable. As Becker (1960) and Franks (1961) point out, to use cortical inhibition, reactive inhibition, satiation, and temporal inhibition, as equivalent terms is not particularly edifying. In addition, Becker, investigating the hypothetical cortical inhibitory process, failed to find support for Eysenck's assumption that satiation and negative drive form a unitary trait, or that a relationship exists between the latter and introversion-extraversion measures.

In recent years, however, (1947, 1953, 1960a, 1960b, 1963) Eysenck has attempted to obtain scientific evidence for the concept of an introversion-extraversion dimension of personality and to relate his experimental findings to the existing body of psychological learning theory by means of a cortical excitation-inhibition construct. This theory, central to the present undertaking is the only known personality theory of its kind. Some of its main characteristics may be reviewed to advantage.

According to Franks (1961), Eysenck's theory is described most often as a "stratification theory", alleged to be made up of four causally related levels. The most fundamental level is the Pavlovian concept of excitation-inhibition balance, held to be a constitutional feature varying with the individual. Depending upon the nature of

this balance, a given individual is assumed to be predisposed to develop more or less excitatory or inhibitory potential (Eysenck, 1960a).

Level two refers to individual differences in performance on a variety of behavioural laboratory tasks, which are assumed to be causally related to individual differences in the excitation-inhibition balance. For example, speed of acquisition and resistance to extinction of CRs are supposed to be facilitated by excitatory potentials and decreased by inhibitory potentials. Similarly, reminiscence is supposed to be a measure of the amount of inhibition generated during massed practice and dissipated during the rest pause. Experimental evidence for the above is to be found in several sources (Eysenck, 1960b, 1963) and will not be reviewed here.

Level three is involved with behaviour patterns observed outside the laboratory; for example, with traits such as impulsivity and sociability, which are subsumed under the general concept of intro-version-extraversion. The link between the personality factors of level three and the experimental phenomena of level two forms the core of Eysenck's system. This relationship, presented first in 1955 states:

> . . . individuals in whom excitatory potentials are generated quickly and strongly, and in whom inhibitory potentials are generated slowly and weakly, will tend to be introverted in personality. Individuals in whom excitatory potentials are generated slowly and weakly, and in whom inhibitory potentials are generated quickly and strongly, will tend to be extraverted in personality. (p. 115)

According to Eysenck, it follows directly from these laws of excitation and inhibition that the introvert will form CRs strongly and quickly, whereas the extravert will form them weakly and slowly.

Level four, that of thought habits and other observable attitudes, is not relevant to the present study.

Theoretical Considerations of the Independent Variables

1. Adaptation and Spence's Theory. As previously described, adaptation refers to that process in which the repeated presentation of a noxious stimulus results in a response decrement. While not explicitly accounted for in Hullian-type inhibition theory, theoretical provision for explaining such a phenomenon is contained within Spence's 1958 theory of emotionally based drive. It is important to note, however, that the predictions made from this theory are related to the concept of drive reduction and not to Hull's inhibition variables.

Spence's theory, one of the few attempts to relate the effects of adaptation to conditioning, is built on Hull's basic assumption that the associate factor (H), and the drive factor (D), combine in a multiplicative manner to determine excitatory strength (E), which in turn determines performance, i.e., $E = H \times D$. It is further assumed that:

> . . . the drive level, D, in the case of aversive situations at least, is a function of the magnitude or strength of a hypothetical response mechanism - a persisting emotional response in the organism, designated as r_e, that is aroused by any form of aversive stimulation. (p. 132)

As a behaviour energizer, this same mechanism is considered to possess properties similar to those of any skeletal response.

On the basis of analogy with observable reflexes to noxious stimuli, several properties are assigned by Spence to the hypothetical response mechanism. The one with which the present study is concerned is based on the adaptive property of observable reflexes; for example, responses that characteristically exhibit adaptation with repeated stimulation:

> On the assumption that our emotional hypothetical response would behave in an analgous manner, it followed that, if a series of trials employing the US alone were given <u>prior</u> to conditioning, a lower level of D would be present during the subsequent conditioning than if no such adaptation trials were given. (p. 132)

From this it follows that if D were lower, a performance decrement would result.

To relate adaptation to Eysenck's theory, it is assumed that this variable involves inhibitory processes. Consequently a series of UCS-alone trials should, through building up temporal inhibition, result in lowered performance during CR acquisition. Eysenck specifically predicts that introverts, in whom excitatory potential is dominant, and in whom negative drive is generated slowly and dissipated quickly, will form CRs with ease. The converse applies to the extravert.

Based on the predictions of Eysenck and Spence, the following hypothesis is stated:

> 1. If, as a consequence of repeated UCS-alone trials, Spence's response mechanism adapts and causes a decrease in drive level; and if further, the same process reduces excitatory potential and increases inhibitory potential, as Eysenck defines these constructs, it is predicted that regardless of ethanol dosage, the acquisition and extinction of the conditioned eyeblink response will be inversely related to the number of UCS-alone trials.

2. Ethanol. Compilation of the multidisciplinary theoretical speculation relative to the effects of ethanol is prohibitive. Whether or not, however, and under what conditions ethanol appears to stimulate or depress certain behaviours seems to be a question of declining importance in the literature. For the purpose of the present study, ethanol, as a central nervous system drug (Goldberg, 1963; Goth, 1963), is considered to have a depressant effect upon performance in a variety of situations. Support for this contention can be found in the reviews of Jellinek and McFarland (1940) and of Walker (1961). The former summarized their conclusions thus:

> Critical analysis of the total evidence of psychological investigations on experimental alcoholic intoxication reveals that alcohol has a depressing effect on all psychological functions yet measured, and that such stimulation as has been reported for some psychological variables is a pseudo-stimulation. . . . (p. 369)

The latter investigator similarly concluded that the vast majority of all experimental evidence shows beyond doubt that ethanol is a depressant drug in its action upon behaviour, "a depressant of all neurophysiological and all psychological activities". When related to the theories of Eysenck and Spence, this independent variable, like the former, leads to a single prediction.

The expansion of Eysenck's excitation-inhibition theory of personality to the field of drug studies is a logical one. As Willet (1963) points out, proceeding from the basic notion that the neural sub-structure of the behavioural dimension of introversion-extraversion is the inhibitory-excitatory balance of the central nervous system, the drug postulate (Eysenck, 1957) states:

> Depressant drugs increase cortical inhibition, decrease cortical excitation, and therefore produce extraverted patterns of behaviour. Stimulant drugs decrease cortical inhibition, increase cortical excitation, and therefore produce introverted patterns of behaviour. (p. 229)

If behaviour patterns resulting from the administration of either stimulant or depressant drugs were found to be concordant with the experimental behaviour patterns of extraverted and introverted groups, the basic Eysenckian hypotheses would be supported. In regard to depressant drugs, Eysenck specifically predicts that eyeblink conditioning will be retarded by their administration due to increased cortical inhibition and decreased cortical excitation.

While Spence does not specifically refer to the effects of drugs upon behaviour, a link can be found in the Manifest Anxiety Scale (MAS), which is regarded as one operational definition of emotional responsiveness (r_e). According to Taylor (1956), the originator of the scale, use of the MAS to select groups that are postulated to differ in drive levels in an experimental situation, rests on the assumption that anxiety scores on the MAS are related to r_e, which in turn effects D. Theoretically, therefore, subjects (Ss) who

differ in anxiety scores on this scale, should manifest corresponding differences in r_e to a noxious stimulus which will result in lowered D and performance. A variety of experiments conducted by Spence and his associates (Baron and Conner, 1960; Spence and Farber, 1954; Spence and Taylor, 1951; Spence and Weyant, 1960; Taylor, 1951) have shown that high anxiety and low anxiety subjects (selected by the MAS) significantly differ as predicted in eyeblink conditioning situations. In the present study, it is expected that ethanol, in its capacity to reduce anxiety, will reduce D and hence CR acquisition.

Predictions derived from the theories of both Eysenck and Spence concerning this independent variable, result in one hypothesis:

> 2. If the depressant drug ethanol increases cortical inhibition, and decreases cortical excitation, as Eysenck maintains; and if further, the same drug produces a reduction in emotional responsiveness which mechanism, according to Spence, directly influences drive level, it is predicted that, regardless of the number of adaptation trials involved, acquisition and extinction of the conditioned eyeblink response will in both cases be inversely related to ethanol dosage.

Although implicit in the foregoing, speculation regarding the relationship between adaptation and ethanol effects leads to a final prediction:

> 3. Since ethanol is presumed to be a central nervous system drug which depresses performance, if adaptation is a similar inhibitory process, it is predicted that acquisition and extinction of the conditioned eyeblink response will be affected similarly by both variables.

CHAPTER 3

Empirical Considerations

Since no specific precedent was found for the present undertaking, this review of the experimental literature will include studies of a more general nature. Topics will be discussed in the following manner: the phenomenon of adaptation, the effects of drugs other than ethanol upon conditioning, the effects of ethanol upon conditioning, depressant drug studies (other than ethanol) related to Eysenck, and finally, a critical analysis of the only reported study in which ethanol was used to test this same theory.

The Phenomenon of Adaptation: As noted in Chapter two, the effect of a noxious UCS upon conditioning has not been investigated as vigorously as other learning variables. True, Spence has theorized about the process of adaptation, but little can be found in the psychological literature that pertains directly to it.

MacDonald (1946) carried out what appears to be the first reported study. Human subjects were used to investigate the effects of adaptation upon both eyelid reflex and finger retraction. In regard to the former, a series of 50 UCS-alone presentations (air puff to the eye) resulted in a marked decrement in CR acquisition during subsequent conditioning trials. Unfortunately, neither the strength of the air puff, nor the rate per minute of the UCS-alone trials (variables themselves having effects upon

the results) were reported. Taylor, however, in 1956 conducted a more sophisticated study and obtained similar results. Each of four groups of subjects received 50 UCS-alone adaptation trials before eyeblink conditioning began. Four levels of UCS intensity were employed. In addition to confirming an hypothesis that one of the functions of the UCS in defence conditioning is to evoke an internal emotional response (Spence's r_e), Taylor found that the strength of r_e, and hence D, was inversely related to the strength of the UCS during adaptation trials.

Indirect information concerning adaptation is to be found in two investigations into the effect of interpolated UCS presentation upon eyeblink conditioning. Dufort and Kimble (1958) and Kimble, Mann and Dufort (1955), reported that UCS-alone trials, interpolated in the middle of a series of CS-UCS conditioning trials, produced a performance increment. Goodrich, Ross and Wagner (1957, 1959) failed to obtain these results and, in fact, reported a response decrement. While this issue is not yet resolved, the variance in these findings, probably attributable to procedural differences, does not detract from the conclusion that UCS-alone trials in some way effect eyeblink conditioning.

An entirely different approach is found in research completed by Jasper (1961). Attempting to get clearer specifications of cortical processes in conditioning, Jasper studied cortical potentials which accompanied conditioned defensive movements in cats. During an initial pre-conditioning adaptation phase, auditory clicks were

presented at a frequency of five per second until the amplitude of the evoked potentials in the auditory cortex had dropped to less than half of the original value. During the following regular conditioning trials, the evoked potentials declined significantly.

The foregoing, however sparse, is believed to be representative of the research conducted upon UCS adaptation process. Hilgard and Marquis (1961, p. 59), commenting upon such sparsity, note also that no systematic study of the effects of different numbers of adaptation trials upon conditioning has been reported.

Effects of drugs other than ethanol upon conditioning:

Although the effect of ethanol upon the conditioned eyeblink response is central to this study, it would be unreasonable to ignore conditioning research that has employed other drugs and other reflexes. This is especially true since psychological investigations into either area are relatively rare (Franks, 1958).

One of the few early studies to be reported was conducted by Skinner and Heron (1937). Twelve white rats and a Skinner box were used to investigate the effects of caffeine (0.10 mg) and benzedrine (0.5 mg) upon the rate of elicitation of a conditioned bar-pressing response. The drugs were given in 0.5 cc of physiological salt solution by subcutaneous injection. It was reported that both drugs increased the rate of elicitation of the CR.

Since 1956, several experimenters have been interested in the effects upon conditioning of such depressant drugs as chlorpromazine,

perphenizine, reserpine and stimulant drugs such as d-amphetamine, benzedrine, caffeine and methylphenidate.

Hunt (1956, 1957) conducted numerous investigations into the effect of meprobamate upon a conditioned emotional response (CER) in rats. In a typical experiment, after training in standard lever-pressing for food, CERs were established by presenting a neutral stimulus (a clicking noise) for several minutes, the termination of which resulted in shock. The experimental animals then received 80 mg/kg body weight of meprobamate. The results consistently showed that this drug did not interfere with the output of lever-pressing.

Brady (1956), employing rats and monkeys in a similar type of experiment, obtained opposite results using reserpine. Both rats and monkeys were trained to bar-press respectively for water and orange juice in modified Skinner boxes. When bar-pressing was stabilized, a CER similar to that used by Hunt was superimposed upon the bar-pressing behaviour. Doses of 1.0, 0.2 and 0.75 mg/kg body weight of reserpine given to the rats, and doses of 0.5, 1.0 and 2.0 mg/kg body weight given to monkeys, resulted in essentially the same results for all animals--a decrease in the behavioural effects of the CER.

In a test of Eysenck's drug postulate, Sinha, Franks and Broadhurst (1958) investigated the effects of both a stimulant and a depressant drug upon a measure of reactive inhibition in 64 rats. Using a simple T maze, it was predicted that the amount of alternation --turning in the opposite direction to that of previous turns--would

be a function of the number of those turns, and further, that this function would itself be increased by amobarbital (15 mg/kg body weight), and decreased by the stimulant drug, pipradol (10 mg/kg body weight). The predictions were confirmed.

Grossman (1961) studied the effects of chlorpromazine and perphenizine on approach-avoidance conflict in rats. After initial training in bar-pressing for food, 24 rats were given shock training until all indicated the presence of a learned fear response to a tone. The rats were then divided into four groups of 6 each, groups 1 and 2 received respectively 0.2 and 0.4 mg/kg body weight of chlorpromazine; groups 3 and 4, 0.125 and 0.250 mg/kg body weight of perphenazine. Both drugs at the different dosage levels reportedly had significant depressant effects.

Hearst (1961), trained 6 albino rats to make two different lever-responses to obtain food and water. When variable-interval response rates had stabilized on both levers, d-amphetamine in doses from 1 mg/kg body weight up to 5 mg/kg body weight was administered. An inverse relationship was found between the dose of the drug and response output.

Bindra and Mendelsen (1962) designed a study to determine if the effects of drugs on a trained response would vary with the habit strength of that response. The effects of two doses of chlorpromazine were determined at several different levels of training of a lever-pressing (water reward) response. Forty-four rats received 2.5 mg/kg body weight of chlorpromazine, and 26, 1.5 mg/kg body weight of the same drug. The mean number of lever presses under

the influence of the drug was studied in relation to training performance. The main findings were: chlorpromazine leads to a decrease in performance, and that the level of drug performance was higher in animals with higher training performance than those with lower training performance.

Mendelsen and Bindra (1962) using 28 rats and "mild" doses of chlorpromazine (1.5 mg/kg body weight) and methylphenidate (4 mg/kg body weight) observed the effects of these drugs upon a conditioned lever-pressing response under different drive levels. They reported that both drugs consistently depressed lever-pressing over a wide range of drive levels. No comment was made at this time about the effects of the stimulant drug. In 1963, however, the same investigators reported that chlorpromazine decreased a conditioned bar-pressing response after a high level of training, (varied number of training sessions) but not at low levels; whereas methylphenidate decreased the rate of the same response at low drive levels, but not at high. Studies such as these emphasize that drug interactions are both a function of the various parameters of the drug used, and of the basic parameters of the behaviour measured.

The effects of ethanol upon conditioning

As indicated above, this section is not intended to be a review of the voluminous literature addressed to the influence of ethanol upon general behaviour phenomena. Such reviews are adequately accomplished by Carpenter (1961), Jellinek and MacFarland (1940),

and Kalant (1962). Unfortunately, however, investigations of the action of ethanol upon conditioning processes, be the subjects animal or human, are both few and limited in their range of applicability. The following studies are considered to be quite representative.

Gantt (1935) was apparently one of the first experimenters to use the CR method to examine the effects of ethanol upon cortical and sub-cortical activity. Doses of 0.5 and 1.5 ml/kg body weight given to five dogs resulted in increased latency of salivary and motor-conditioned responses. Larger doses (3.0 and 4.0 ml/kg body weight) decreased the intensity of the CR. Dworkin, Bourne and Raginsky (1937) extended Gantt's work on conditioned salivary responses to conditioned alimentary-motor responses in dogs and cats. Doses of 25 to 35 mg/kg body weight of amytal, and doses of 3.0 to 3.5 ml/kg body weight of ethanol depressed the conditioned response.

Sokolova (1954), again investigating the effects of ethanol upon the conditioned salivation response in dogs, found that ethanol in doses 0.25 to 0.5 ml/kg body weight speeded up the processes of conditioned inhibition as measured by extinction trials.

A different approach was used by Settlage (1936). In an investigation of the effects of sodium amytal upon the formation and extinction of the CR, ethanol and nembutal were used for comparative purposes. It was reported that the effects of all three drugs upon 12 cats were to inhibit the processes underlying the elicitation of the CR.

Depressant drug studies (other than ethanol) related to Eysenck

This section will be concerned with reporting and commenting upon depressant drug studies designed to test Eysenck's drug postulate. It will be recalled that this is based on the belief that introversion-extraversion and conditionability[1] are related to the cortical processes of excitation and inhibition, as originally conceived by Pavlov. Accordingly, a drug which increases cortical inhibition should, by decreasing condition-ability, result in a CR decrement. Using eyeblink conditioning techniques, Eysenck and his co-workers have tested such predictions with considerable success. Detailed reviews of these studies were made in 1963 by Trouton and Eysenck. Since the general procedures and apparatus are standardized, only one or two examples will be cited.

Franks and Laverty (1955) investigated the effects of sodium amytal upon eyeblink conditioning. The Ss were 16 hospitalized neurotics, all diagnosed as dysthymics, and thus according to Eysenckian theory, expected to condition readily. Four treatments were employed: a large dose of sodium amytal, 0.05 Gm/kg body weight, and a smaller dose always 0.01 Gm less than the larger dose would be for each S; one placebo and one no drug. Conditioning took place in a soundproof room, the UCS was a puff of air delivered to one eye at a pressure of 65 mm of mercury, and lasting for 500 msec. The CS was a pure tone of frequency 1100 cycles per second.

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[1]Conditionability is a personality variable suggesting individual differences in rate of conditioning.

It was reported that intravenous sodium amytal reduced the number of conditioned eyeblink responses during acquisition, and increased the rate of extinction.

Using the same equipment, Franks and Trouton (1958) studied the effects of amobarbital sodium and d-amphetamine upon the same process. The aim of the experiment was to compare the acquisition and extinction of the eyeblink CRs while under the influence of a stimulant or depressant drug. Eighty female Ss received at random one of four treatments: amobarbital--4.5 mg, d-amphetamine --10 mg, 45 minutes before conditioning, or d-amphetamine two hours before conditioning, and placebo. It was found that the two hour d-amphetamine group conditioned more readily than the placebo group, whereas the amobarbital group conditioned less readily.

Ludvigson (1964), in a criticism of the two studies above-mentioned, noted that relatively few acquisition trials were given, and that the use of test trials resulted in conditions of partial reinforcement. A study, using three groups of 20, 23 and 20 volunteer males, receiving respectively chlorpromazine, d-amphetamine and a placebo, was conducted to investigate the effects of 80 consecutive reinforced trials upon conditioned eyeblink response acquisition. All Ss took one tablet five hours, and another tablet one hour, prior to the conditioning session. Since the chlorpromazine tablets were 25 mg, and the d-amphetamine were 5 mg, the total doses were 50 mg and 10 mg respectively. The results indicated no

significant differences between the drug groups, leading Ludvigson to suggest that these drugs affect, not conditioning as usually defined, but rather sensitization, a phenomenon which the test trial procedure of the previous studies might reveal, if examined.

Willet (1963) conducted a study which appeared to have particular interest to the present undertaking since the declared intention was to examine the relationship of inhibitory tendencies to eyeblink conditioning. The Ss were 24 normal adults who received on different days Doriden, meprobamate or no drugs. Two 250 mg tablets of Doriden were given, one in the morning when the S arrived at the laboratory, and the other four hours later. The same procedure was followed with the meprobamate, the dose being two 400 mg tablets. In the case of both drugs a period of 45 minutes was allowed to elapse between administration and the commencement of testing. The significance of the principle findings was complicated by the fact that while Doriden resulted in a clear depression of the conditioned eyeblink response, meprobamate did not. Willet attributed the latter result to inadequate dosage.

The foregoing, typical of the work conducted by the Eysenckian school, in general finds support for Eysenck's theory.

Ethanol and the conditioned eyeblink, a study by C. M. Franks (1963)

A separate section is reserved for Franks' research for two reasons: in addition to being the only known study to use ethanol to test Eysenck's drug postulate, it is one of extraordinarily few attempts to investigate the relationship between ethanol and

human conditioning. As stated in Chapter two, although the need for such undertakings is frequently expressed in the literature, experimenters appear to be reluctant to involve themselves.

Franks (1963) designed his study to test as completely as possible the many facets of Eysenckian theory. Eighty-eight adult normal males were randomly assigned to control and experimental groups. Before and after drinking the treatment beverage, both groups were subjected to a battery of tests, which included eyeblink conditioning. The remarks that follow are restricted to this one aspect. The control group received an insignificant amount of ethanol (unrecordable on the drunkometer), and the experimental group received 1 ml of 90 proof blended whiskey per kg body weight. Blood alcohol levels were assessed repeatedly with the Harger Drunkometer. Contrary to all expectations, ethanol failed to inhibit the acquisition and resistance to extinction of the conditioned eyeblink response. These negative results may, in part, be attributable to uncontrolled variables; variables to which the present study has addressed itself most carefully. Since these are dealt with at some length in Chapter four of the present thesis, only brief reference will be made to them at this time.

Although the Ss in Franks' study were cautioned not to eat or drink prior to the experimental session, the adequacy of this method alone to control for stomach content may seriously be doubted (Goldberg, 1963). It is possible, therefore, that blood alcohol

concentrations were considerably affected. It is noted also that the Ss were allowed to consume the beverage at their own rate. The average time to consume the drink was 26 minutes, but the range extended from 3 to 60 minutes. Lack of control over this variable must inevitably effect the absorption rate of the ethanol (Carpenter, 1961; Drew, 1963; Goldberg, 1963) and the blood alcohol concentration at any locus, again leading to possible error in interpretation. Similar error may also be attributed to the large discrepancies among Ss in the length of time that was allowed to elapse between finishing the drink and taking the first blood alcohol level reading; for example, subject one, 60 minutes; subject seven, 75 minutes; subject fifteen, 90 minutes; subject twenty-four, 42 minutes; and subject thirty-four, 73 minutes.

A criticism of a different nature is related to the intensity of the UCS. It is reported that the corneal air puff was delivered at a pressure of 170 mm. From a general review of the literature, this might be regarded as being a fairly strong noxious stimulus. While there are exceptions, it would seem that a majority of experimenters (including Franks himself on other occasions) use pressures ranging from 50 - 100 mm, with a mean around 70 mm. Although in most investigations of the human cornea to air stimuli, ethanol has been shown to depress the sensitivity to touch or pain stimuli (Goldberg, 1963), there is reason to suspect that the degree of anesthesia attained by the average blood alcohol

concentration of 0.09 reported by Franks, might be insufficient to counter the noxiousness of the UCS. As a consequence, CRs, which are nothing more than anticipatory responses, would not necessarily diminish.

CHAPTER 4

Method

Selection of the Sample

Subjects were 135 non-abstaining males, aged 21 or over, all volunteers from the University of Alberta student population. Psychology students were excluded from the study in view of the probability that they would have knowledge of conditioning theory. All Ss were apparently free from mental and physical illness, and from drug addiction and alcoholism. Remuneration was at the rate of $1.25 an hour. An additional 20 Ss were discarded for the following reasons: 10 became nauseated and were unable to complete the experiment; 5 inadvertently received the wrong doses of ethanol; the results of 3 others were spoilt due to equipment breakdown; 2 were discovered to be heavy drinkers of some years standing; and one pseudo-conditioned.

Apparatus

The eyeblink conditioning equipment was located in a suite of two rooms in the University of Alberta psychological laboratory. The Ss' room was sound-treated and consisted of an enclosure in which the S sat on a straight-back chair facing into a flat white and well-illuminated area. Room temperature was maintained at 70° F. The S wore a headband supporting an air jet with a 2 mm orifice directed at an upward angle, and at a distance of approximately 3/8 in. from the right cornea. A 54 msec puff of compressed

air served as the UCS, which was of sufficient intensity at the eye to support an 80 mm column of mercury. The CS, a tone, was delivered to both ears through a standard head set. The headband also supported a microtorque potentiometer coupled to the S's right upper eyelid by a light mechanical linkage. Movement of the lid produced a signal from the potentiometer which was amplified by a Brush DC amplifier and recorded by means of ink-writing brush pen motor. A Western Union tape transmitter controlled the inter-trial intervals and activated electronic timers controlling the duration of the CS - 560 msec, and of the 506 msec CS-UCS interval. The experimenter and the recording and stimulus programming equipment were housed in a room adjacent to the conditioning room.

Blood alcohol readings were taken on the Borkenstein breath-alyzer. The rationale underlying its use is based on the fact that a small proportion of ethanol which is absorbed into the blood is excreted in the breath. Alcohol in alveolar air is in equilibrium with that in pulmonary capillary blood. The ratio 1:2,100 between blood and deep lung air allows the results of breath analysis to be used as an indicator of blood alcohol levels (Begg, Hill and Nichols, 1964). Thus deep lung air is collected in the breathalyzer which measures a given volume at a fixed temperature, above the temperature of breath, bubbles it through a 0.025% solution of potassium dichromate in 50% sulphuric acid, and by means of a built-in colorimeter with twin cells and a null-reading galvanometer measures the degree of reduction of the dichromate by the ethanol. The result is obtained directly on a scale in terms of blood alcohol concentration.

Design

A 3 x 3 factorial design involving three dosages of ethanol and three levels of adaptation was used. Ss were randomly assigned to one of the three ethanol groups, and to one of the three adaptation groups. Random assignment to the time of day of experimental session was not wholly achievable due to lengthy post-experimental recovery of the high blood alcohol level group. During the study, however, approximately an equal number of Ss from each of the nine conditions were tested morning, afternoon and evening, throughout any given week.

The blood alcohol levels (mg/100 ml) it was hoped to achieve for the three groups were: 0.00[1], 0.05, and 0.10. The choice of such blood alcohol levels, far from being arbitrary, was worked out in terms of the quantity of ethanol needed to attain them, individual body weight, and the ethanol content of the beverage. In the case of the highest concentration, for example, the question to be answered was how much ethanol can a fairly inexperienced S drink within a 20 minute period without becoming ill or impaired to the point of uselessness.

The calculations used were: for the blood alcohol level 0.05 group, 0.85 ml of 70 proof whiskey per kg of body weight; for blood alcohol level .10 group, 1.7 ml/kg of body weight. Each dose was

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[1]Blood alcohol level 0.00 refers to placebo groups which did not receive any ethanol. This terminology is maintained throughout the study for ease of reporting.

given to the S as a mixture of whiskey and iced ginger ale in a 1:2 ratio. The placebo group received 5 ml/kg body weight of iced ginger-ale only.

The three levels of adaptation were achieved by using the UCS, a puff of air delivered to the cornea, in the absence of the CS at the rate of 5 trials a minute as follows: no trials, 35 trials, and 70 trials.

Procedure

Although the conditioning procedure was central to the study, approximately 70% of the S's laboratory time was spent in preparation for this experience. This is referred to as the "pre-test phase", the test in turn referring to the actual conditioning. The pre-test phase, which took precisely 90 minutes, was primarily concerned with the administration of the appropriate doses of treatment beverage, and with control of the time factor involved in reaching a predetermined blood alcohol level. The total time of S involvement was two and one-half hours.

The major problems that had to be resolved emanated mainly from the physiological variables involved in the fate of ethanol once ingested. For example, the amount of ethanol found in the human body after drinking is the result of an interplay between several processes: the rate of absorption of the ethanol from the gastrointestinal tract, its distribution by the circulating blood to various organs and diffusion into body fluids; body weight and the percentage of total body water, and finally, the rate of

disappearance of ethanol from the body by excretion and metabolic breakdown (Ferguson and Bell, 1955; Goldberg, 1963; Goth, 1961; Wayne, 1963). Attempts to control for as many as possible of these variables, and thereby blood alcohol levels, are now described.

The rate at which ethanol is absorbed into the blood stream depends upon several factors. The most obvious, and the easiest with which to contend, is the ethanol content of the beverage used. Throughout the present study, Seagram's 3-star 70-proof rye whiskey was used.

The presence or absence of other substances in the gastrointestinal tract at the time ethanol is ingested is also critical to the absorption rate. It is well established (Kalant, 1961; Loomis, 1950) that ethanol is absorbed more rapidly on an empty stomach, under which conditions the average maximum blood alcohol level is reached much sooner than if the same quantity were ingested on a full, or partially filled, stomach. Control for this potentially large source of variation was achieved by insisting that all Ss neither ate nor drank any substance for at least two hours prior to the experimental session. Additionally, a "stomach-emptying" technique, empirically validated by the members of the University of Alberta Pharmacology Department, was employed as an aid to controlling for the ethanol absorption rate. Upon arrival at the laboratory, the S was given 500 cc of tap-cold water. Five minutes were allowed for its consumption, and a further 25 minutes for the process to take place before the treatment beverage was administered.

The rate of disappearance of ethanol from the blood was considered in relation both to dosage and time. In the case of the former, it was found that the rate of disappearance is relatively constant and independent of dosage (Goldberg, 1963; Goth, 1961). In regard to the latter, the concern over time arose upon realization that the different number of UCS-alone adaptation trials (zero, 35 and 70) would differentially increase the length of stay in the conditioning room for the three ethanol groups. As it was the intention to average the blood alcohol level readings taken before and after conditioning in order to arrive at one reportable blood alcohol level, it was thought possible that the rate of disappearance of ethanol from the blood might operate as an uncontrolled variable. Turning to the literature again (Coldwell and Smith, 1959), it was found that the disappearance of ethanol occurs approximately at a rate of 88 mg per kg body weight per hour. Since 50 minutes was the longest expected time in the conditioning room for any S, it was considered that biological variation due to this factor would be of no significance.

The final variable to be noted in this context is drinking experience. This was not thought likely to be of any concern in view of the population sampled. Since, however, evidence exists (Goldberg, 1943) to show that the relationship between blood alcohol level and observed change in performance is partly a function of drinking experience, each S was asked to indicate the amount of ethanol, in whatever form, he customarily consumed (see Appendix C). With two exceptions it was possible to classify all subjects as "drinkers of limited experience".

Two additional problems of experimental design, related but of a different nature, had also to be accommodated: invention of an acceptable placebo, and control over pre-test time spent in the laboratory by Ss in different ethanol dosage groups.

In regard to the former, credit for the idea of the type of placebo must go to Franks, 1963. As this investigator suggested, capsules, tubes, and disguises such as peppermint oil have generally been unsuccessful in ethanol studies with human Ss. With minor modifications, Franks' own solution to this problem was utilized in the present experiment. The placebo beverage was contained in an amber glass around which was placed a woollen cozy, liberally soaked in rye whiskey. The rim of the glass, and the handle of the container holding the balance of the fluid, was similarly doctored. The efficacy of this procedure was most gratifying. A research worker, independent of the author, who casually questioned each placebo S at the end of the session, reported no case in which it had been doubted that "some" ethanol had been given.

The control of pre-test time was necessary in light of empirical evidence that Ss receiving the maximum dose of ethanol required 60 minutes from commencement of drinking to attain the prescribed blood alcohol level. Although the blood alcohol level 0.00 group was not thus involved, experimental design demanded that all three ethanol groups be subjected to standardized pre-test phase procedures in terms of time and activity. Accordingly, all Ss, as previously

mentioned, were involved in a 30 minute "stomach emptying" process. This time was also utilized to read the instructions to the S, to acquaint him in general terms with the nature of the experiment, and to familiarize him with the breathalyzer, an actual sample of exhaled air being taken. This sample (blood alcohol level 1), was analyzed in the S's absence (as were all subsequent samples) as a precautionary measure against acquiring Ss who may have been drinking prior to arrival.

Following this, the appropriate treatment beverage was given, the S being encouraged to consume it within 20 minutes[1]. Blood alcohol level 2 was taken 40 minutes from the time drinking began; blood alcohol level 3 sixty minutes from commencement, and immediately prior to the S being taken into the adjacent conditioning room. During the 90 minute pre-test period, Ss were encouraged to relax, and to occupy themselves with such facilities as taped music, radio, playing cards, reading matter, and 16 mm short films.

Once in the conditioning room, the S was seated, attached to the conditioning equipment, and administered UCS-alone adaptation trials according to assignment. Without pause, conditioning proper began, each S receiving 100 acquisition trials and 10 extinction trials with randomly distributed inter-trial intervals of 15, 20 and 25 seconds with a mean of 20 seconds. Any deflection from

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[1]Mean drinking time for all groups, 16 minutes. Range, 15-21 minutes.

the base-line of at least one mm occurring between 150 and 500 msecs was scored as a CR. Extinction trials were of a non-reinforced UCS-omission type.

At the conclusion of this procedure, blood alcohol level 4 was taken. Those Ss who received either the placebo or the small ethanol dose, were then allowed to leave the laboratory. The Ss in blood alcohol level 0.10 group were detained for an appropriate length of time.

CHAPTER 5

Results

The average blood alcohol level for the two ethanol groups, taken before and after conditioning, were: blood alcohol level 0.05 group, $\bar{X}$ = 0.049, SD = 0.004; blood alcohol level 0.10 group, $\bar{X}$ = 0.099, SD = 0.015. Table 1 indicates the blood alcohol levels attained by both ethanol groups before and after conditioning.

TABLE 1

Means and standard deviations of blood alcohol levels for both alcohol groups, taken before and after conditioning

Blood Alcohol Levels (mg/100ml)	Mean before Condi-tioning	Standard Devia-tion	Mean after Condi-tioning	Standard Devia-tion
0.05	0.052	0.0001	0.047	0.0001
0.10	0.103	0.0104	0.099	0.010

Conditioned response acquisition curves for the nine groups (n = 15) are shown in Figs. 1, 2 and 3, from which it would appear that both ethanol and adaptation depressed CR acquisition. That interaction is minimal among these groups is shown in Fig. 4 which shows total CRs as a function of adaptation trials. Analysis of

variance of total CRs, in the form of a 3 x 3 factorial design, Table 2, confirms that both the effects of the three doses of ethanol (A effects), and the three levels of adaptation (B effects), are significant at the .01 level, and further, that A x B interaction is not significant. It is shown in the same table that the A and B effects, while linearly significant at the .01 level, were not quadratically significant.

In regard to the extinction data, no significant differences exist in any respect. Out of 135 Ss, giving a possibility of 1,350 CRs during extinction, only 119 such responses were recorded, and these were randomly scattered throughout the nine groups.

As shown in Fig. 5, ethanol and adaptation had depressive effects upon the amplitude of the first UCR elicited under conditioning procedures. Table 3, an analysis of variance in the form of a 3 x 3 factorial design, confirms that the A effects are significant at the .05 level, the B effects at the .01 level, and that A x B interaction is not significant. Further, that the A effects are significantly linear at the .05 level, and the B effects at the .10 level. In neither case is there quadratic significance. This analysis was undertaken to discover how each independent variable effected the magnitude of the UCR. Such a procedure was invoked in light of evidence which has shown that the level of conditioning may be related to the magnitude of the UCR (Hilgard, 1961).

In Figs. 6 and 7, the CR latency frequency distribution of the three adaptation groups (no ethanol, n = 15), is compared with the frequency distribution of the three ethanol groups (no adaptation, n = 15). While showing a general increase in time as a function of ethanol dosage and adaptation levels, the similarity of the modes and distribution shapes suggests that CR latency does not differ significantly for either variable. Statistical analysis of this data was impossible due to the large irregularity in the numbers of responses given by individual Ss. CR latency medians, however, are given in Table 4. No systematic differences are apparent.

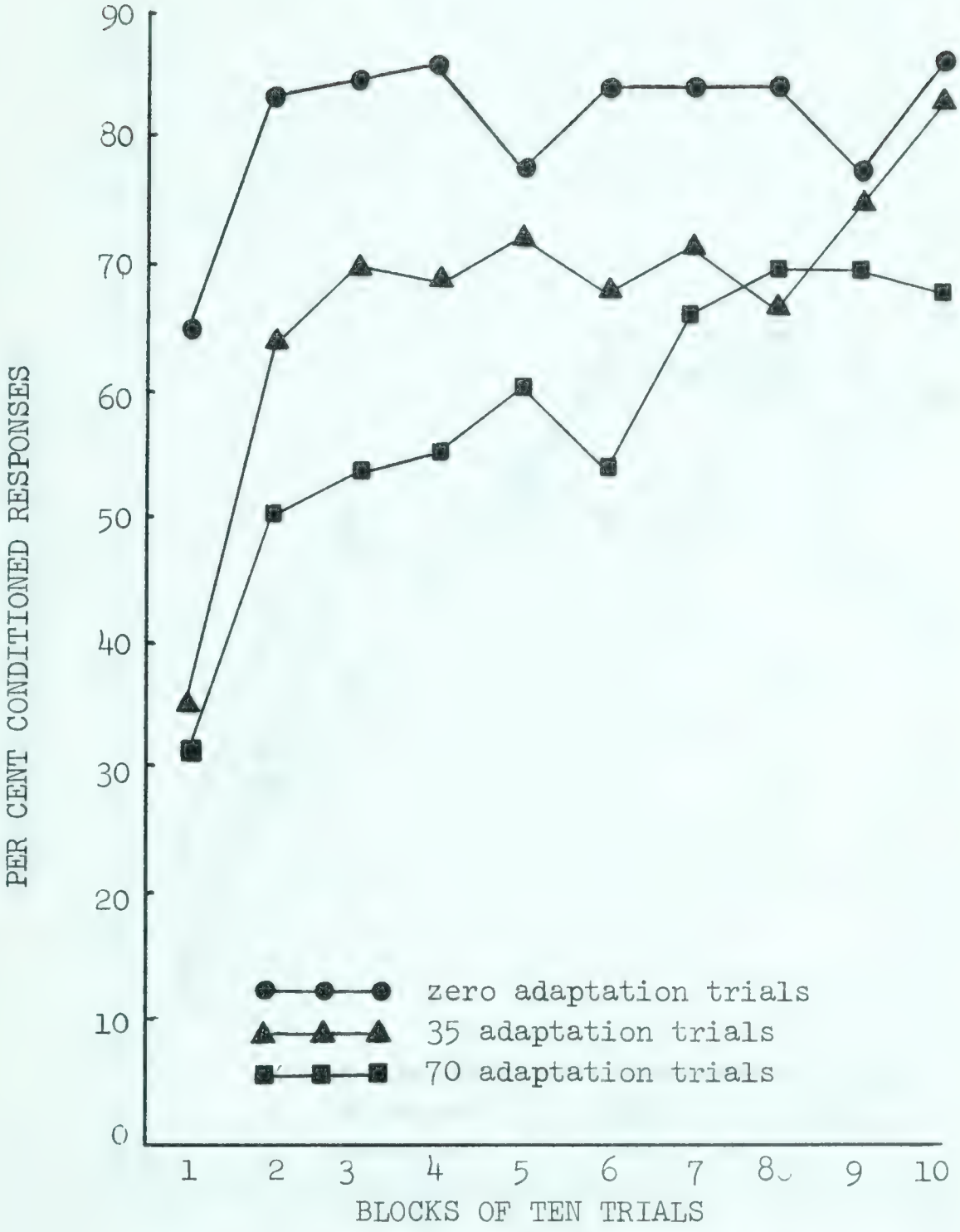

Fig. 1. Percentage of conditioned eyeblink responses for blood alcohol group 0.00, shown as a function of zero, 35 and 70 adaptation trials.

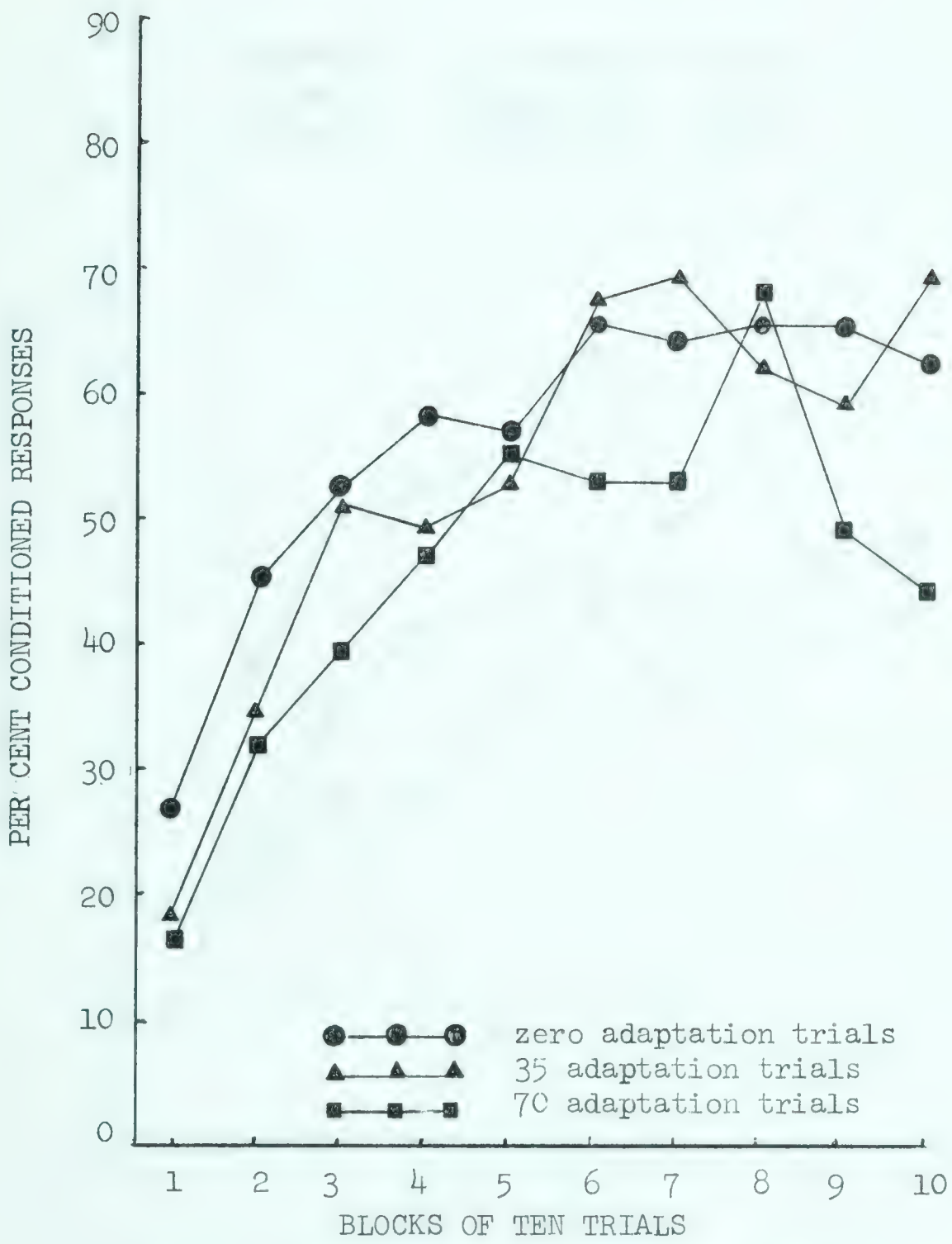

Fig. 2. Percentage of conditioned eyeblink responses for blood alcohol group 0.05, shown as a function of zero, 35 and 70 adaptation trials.

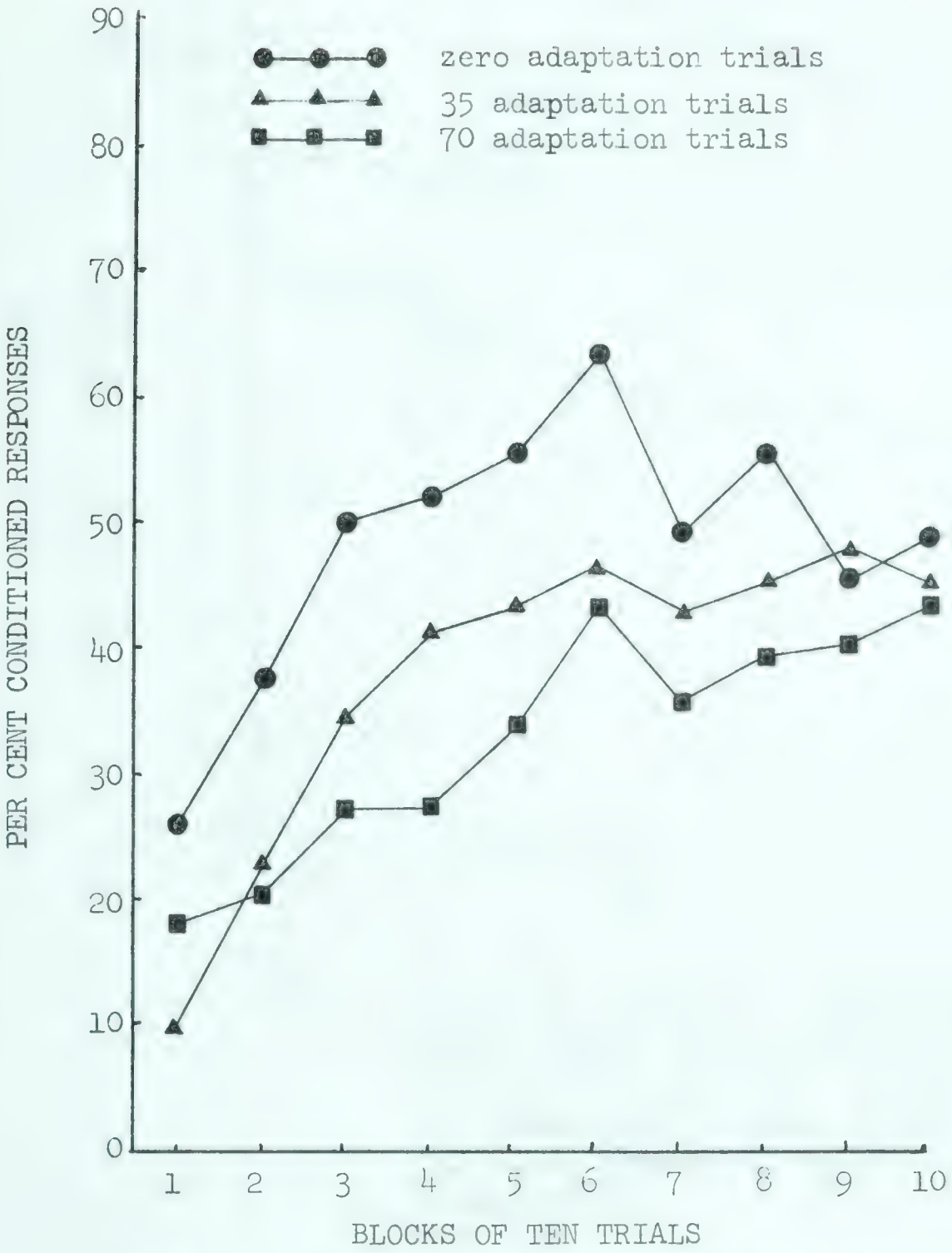

Fig. 3. Percentage of conditioned eyeblink responses for blood alcohol group 0.10, shown as a function of zero, 35 and 70 adaptation trials.

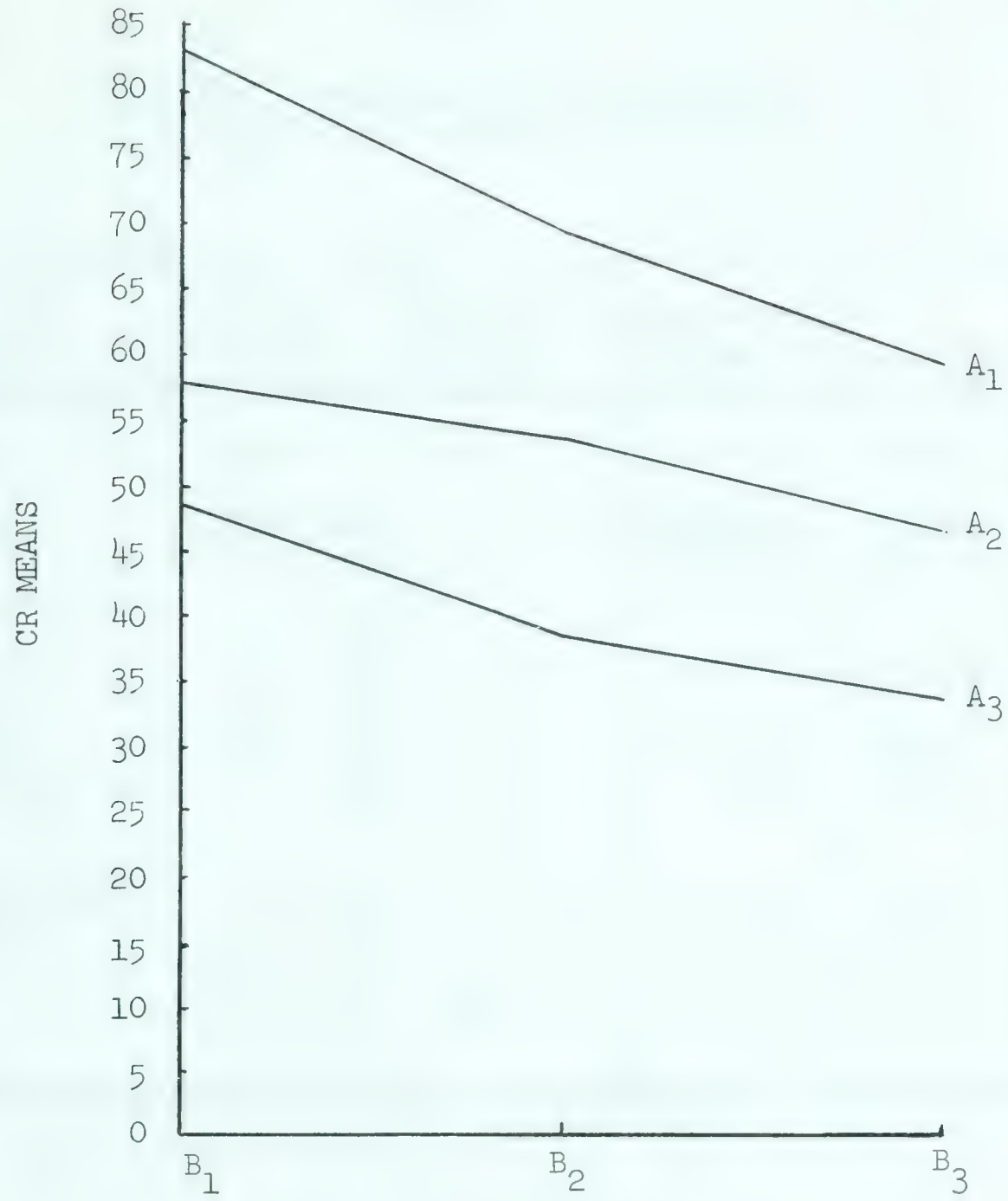

Fig. 4. Means for levels of A at each level of B. A_1, A_2 and A_3, correspond to blood alcohol levels of 0.00, 0.05 and 0.10 respectively. B_1, B_2 and B_3, correspond to zero, 35 and 70 UCS-alone adaptation trials, respectively.

TABLE 2

Analysis of Variance of CR acquisition
for nine treatment groups

Source of Variation	Sum of Squares	Degrees of Freedom	Mean sum of Squares	F
Ethanol	19320.934	2	9660.467	17.33*
Linear	14288.400	1	14288.400	25.62*
Quadratic	46.450	1	46.450	NS
Adaptation	6035.244	2	3017.622	5.412*
Linear	6035.210	1	6035.210	10.82*
Quadratic	.033	1	.033	NS
Ethanol x Adaptation	700.889	4	175.222	NS
Error	70254.667	126	557.57	-

* p = .01.

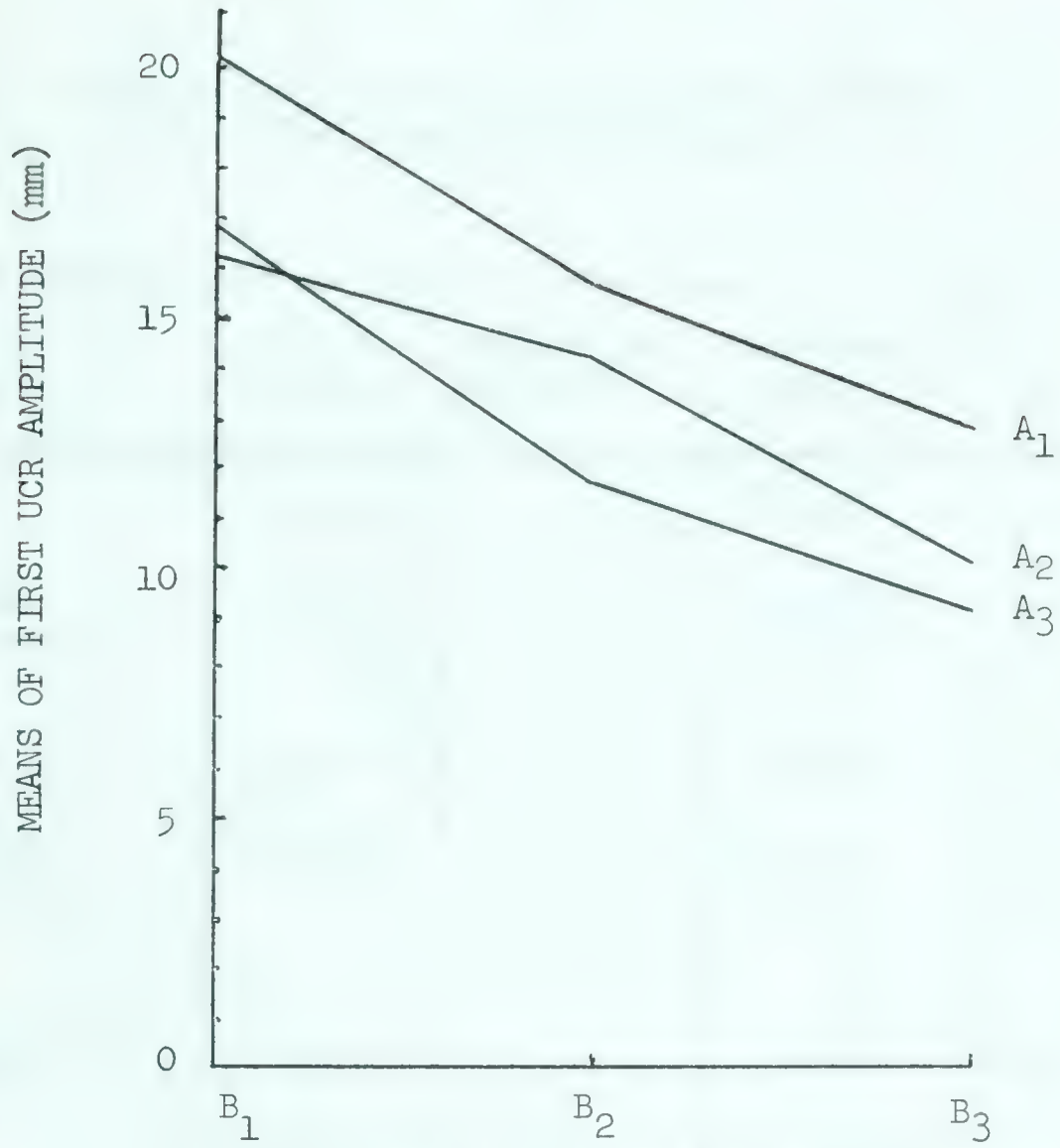

Fig. 5. Means of first UCR amplitude for levels of A at each level of B. A_1, A_2 and A_3 correspond to blood alcohol levels of 0.00, 0.05 and 0.10, respectively. B_1, B_2 and B_3, correspond to zero, 35 and 70 UCS-alone adaptation trials, respectively.

TABLE 3

Analysis of variance of first UCR amplitude
for nine treatment groups

Source of Variation	Sum of Squares	Degrees of Freedom	Mean sum of Squares	F
Ethanol	337.29	2	168.645	3.25**
Linear	313.60	1	313.60	6.04**
Quadratic	23.69	1	23.69	n.s.
Adaptation	1112.020	2	556.010	10.70*
Linear	1109.50	1	1109.50	21.37*
Quadratic	2.50	1	2.50	n.s.
Ethanol x Adaptation	46.44	4	11.61	n.s.
Error	6541.29	126	51.915	-

** p = .05

* p = .10

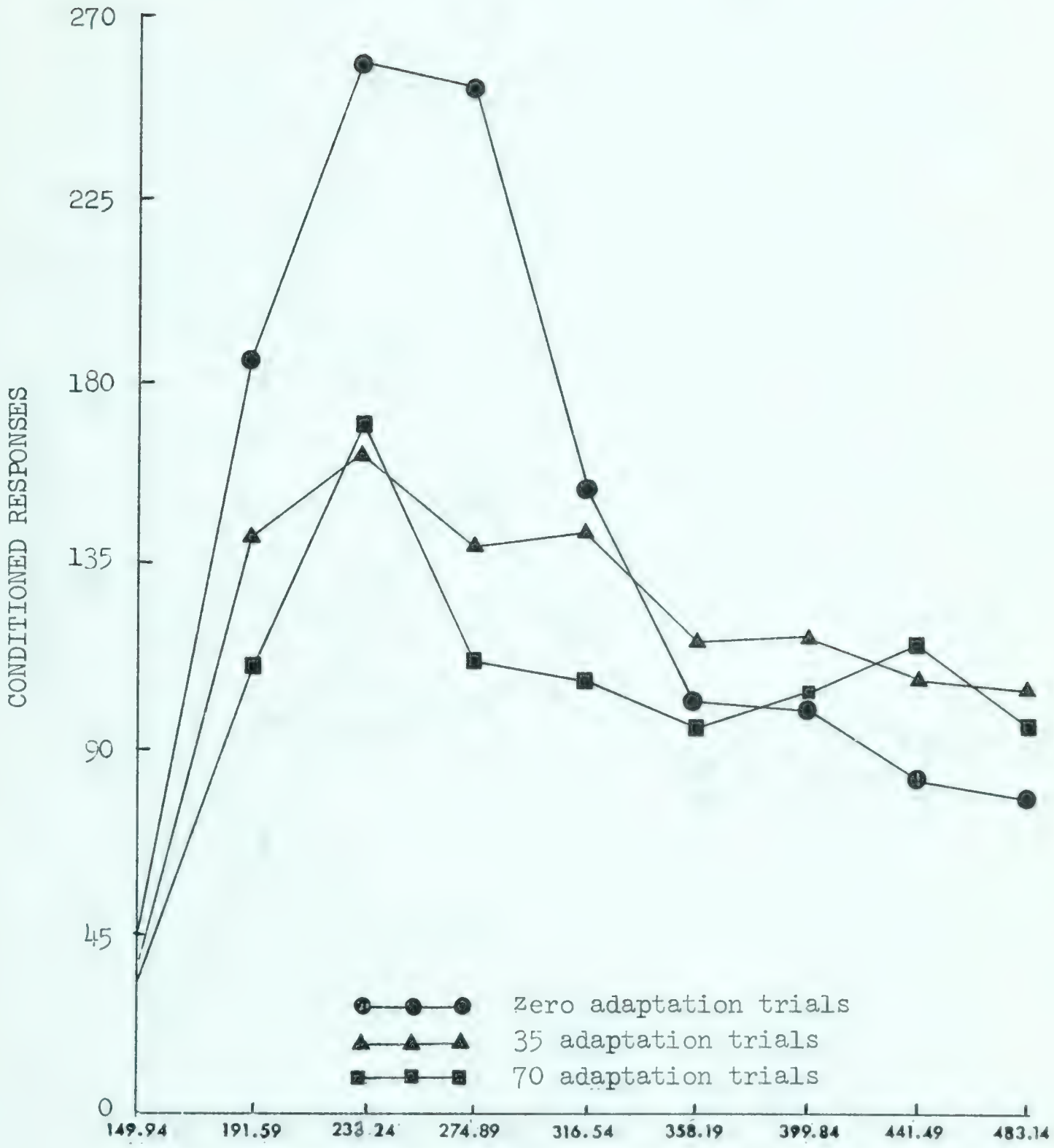

Fig. 6. Frequency distribution of conditioned eyeblink response latencies for three ethanol groups (n = 15 per group) receiving adaptation trials but no ethanol.

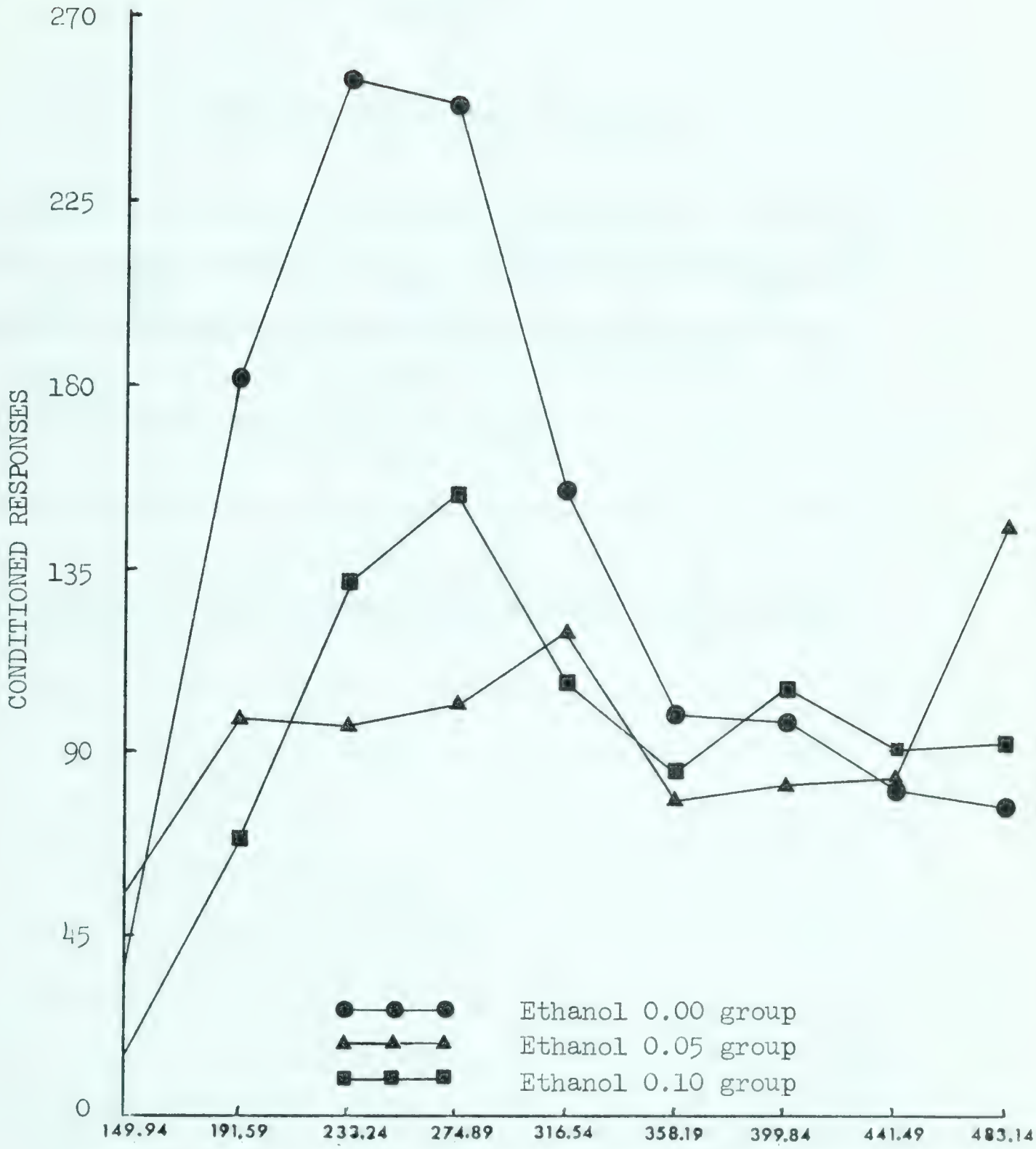

Fig. 7. Frequency distribution of conditioned eyeblink response latencies for three ethanol groups (n = 15 per group) receiving ethanol but no adaptation trials.

TABLE 4

Median CR latency for all groups

Blood Alcohol Levels (gm/100ml).	Adaptation trials		
	Zero	35	70
0.00	35.5	53.2	51.2
0.05	50.8	54.2	63.7
0.10	56.3	63.2	61.9

CHAPTER 7

Discussion

The main findings of this experiment were that the effects of ethanol and of adaptation upon the acquisition of the conditioned eyeblink response were both similar and independent. The implications of these results for the theories of Eysenck and Spence will next be examined.

Ethanol: the hypothesis derived from Eysenck's drug postulate was confirmed. As predicted, CR acquisition was found to be inversely related to ethanol dosage. If Eysenck's theory is valid, such findings may be interpreted to support the contention that the action of ethanol is to increase inhibitory potential, decrease excitatory potential, and to produce "extraverted" behaviour. These results are contradictory to those obtained by Franks (1963). It is believed that the present study is the first of its kind to investigate Eysenck's personality theory, and the first to find support for it using the depressant drug ethanol.

In regard to Spence's theory, if the effects of the three ethanol doses, from small to large, are conceptualized to be reducers of emotional responsiveness, in turn leading to three levels of D, ethanol had the predicted effect-which was to reduce D and thus CR acquisition. Such results appear to provide evidence to support Spence's theory of emotionally based drive. As far as can be ascertained, this study represents the first application of such methodology for this purpose.

Adaptation: As Spence predicts, adaptation of r_e to repeated UCS-alone trials produced a CR decrement. These results, which most clearly support Spence's theory, are in accord with the findings of MacDonald (1946) and Taylor (1956).

Within Eysenck's theoretical structure, if adaptation is assumed to be a form of I_R, its action predictably resulted in "extraverted behaviour". This again may be taken as evidence in favour of the theory that conditionability is related to a cortical excitation-inhibition balance. This is the first time, as far as can be ascertained, that this method of manipulating inhibition has been employed to test Eysenck's theory.

Finally, the hypothesis which led to the prediction that the effects of ethanol and of adaptation were similar cortical inhibitory processes, was confirmed. The implications of this finding are discussed below.

General Discussion

The most thought-provoking aspect of the results of this research lies in the non-significant interaction between the A and B effects, both in respect to CR acquisition and to UCR amplitude. The significant linear relationship of both independent variables may be interpreted in two ways: it can either be hypothesized that each variable affects performance independently and through its own mechanism, or, that only one mechanism is involved, and that the affects of both variables are equivalent and additive.

From Eysenck's theoretical standpoint, either of these views could be maintained. In the first case, it might be suggested that while both variables act as inhibitors of cortical excitation, they do so at different loci and may therefore be expected to produce independent but similar results. It might further be suggested that, whereas ethanol increases cortical inhibition, adaptation reduces D directly as a form of I_R, perhaps involving the reticular formation. This is the first hint of the possibility of two separate inhibitory mechanisms.

If only one mechanism were postulated to be involved, ethanol, as a depressant drug, would be hypothesized by Eysenck to have acted as a cortical inhibitor to produce negative drive and CR decrement. Adaptation would be attributed also to "central" negative drive, the effects of both variables being additive.

In either of the above cases, however, Eysenck would have difficulty explaining UCR amplitude decrement where ethanol alone was involved, since I_R cannot build up unless a response is made. One way out of this dilemma, however unlikely, would be to say that ethanol in some way produces I_R.

In terms of Spence's theory, while it would be no problem to account for the UCR amplitude results, only one interpretation of the principle findings of this study appears possible. This interpretation, restricted to a one-mechanism concept, would have to be based on the assumption that the adaptation function is a linear one. In this event, interaction between the A and B effects

would not be predicted, thus the effects of both variables would have to be equivalent and additive, and act similarly upon r_e to reduce D. That this assumption of linearity has to be made becomes clearly evident when the consequences of the alternative assumption --that the adaptation function is negatively accelerated--are examined. In this event, an A x B interaction would have to be predicted. It would then be impossible to claim that both ethanol, as a reducer of anxiety (and thus of D), and UCS adaptation of the r_e, independently produce the same effect. Committed as he is to a theory of emotionally based drive, the lack of a relationship between the effects of ethanol and of adaptation would be difficult, if not impossible, for Spence to explain.

The former explanation based on the assumption that the nature of the adaptation function is linear is plausible, but its adequacy can be doubted. It is difficult to concede that no mechanism other than r_e is involved in the adaptation process. Spence's own inconsistency in regard to the effect of the UCS on drive level reinforces this doubt. Repeated UCS-alone trials are held to reduce D through the r_e mechanism, yet at the same time the UCS is claimed to maintain D during conditioning.

At a more general level, the lack of interaction between ethanol and adaptation appears to violate commonsense. It would normally be expected that a person with a blood alcohol level of 0.10 would be far less sensitive to the UCS than one who has not had ethanol, yet the results indicate that ethanol had no significant effect upon the reaction of the Ss to the adaptation procedure.

This might be explained by assuming that both variables inhibit performance in the same general way, but either through different cortical mechanisms, or through selective inhibition of the same mechanism. Some support for either speculation, while anything but conclusive, can be extracted from two opposing views related to the locus which ethanol is held to act upon.

The first involves the classical explanation of the effects of ethanol upon the cortex (King, 1957; Walker, 1961). In this view, the several levels of brain structure--cerebral cortex, mid-brain, hind-brain, and spinal chord--are believed to represent progressively lower levels of structure and function. It is assumed that, since each level is under the control of the higher level, the effect of ethanol would be to depress the various levels in order. If this view were to be maintained, it might be hypothesized that, while ethanol inhibits the cortex in the classical manner, the adaptation process involves the reticular formation.

An alternative explanation results from the work of such as Caspers (1958), Goldberg (1943) and Morruzzi and Magoun (1949), whose research casts doubt upon the adequacy of the classical explanation.

Goldberg, for example, hypothesized that if the classical view were valid, intellectual processes should be impaired at lower blood alcohol levels than motor or sensory processes. The results of a well controlled experiment indicated that intellectual

functions (problem solving) returned to normal sooner at higher blood alcohol levels than motor or sensory functions. Such results are at variance with the predictions that classical theory would make.

Supporting evidence of a different nature comes from Casper's study. Using ethanol and implanted micro and semi-micro electrodes in conscious, unanaesthetized rats, he reported that spontaneous electrical activity of the cortex was directly influenced by the level of activity in the reticular formation. Depression of the latter by ethanol was found to relax the cortex in a manner described as "not serving any function". Carpenter (1961), Kalant (1962), and others have interpreted such results to mean that ethanol is not primarily a cerebral cortical depressant, but a selective inhibitor of some part of the reticular formation.

On the basis of the above, lack of A x B interaction might be accounted for by assuming that the action of both variables is selective upon the reticular structure. How this would occur must remain purely speculative. Inhibitory modification of afferent activities appears to be accomplished by both ethanol and the process of adaptation--perhaps selectively because some nerve pathways, while sensitive to the effects of ethanol, are resistant to the effects of adaptation, and vice versa.

Summary of the foregoing leads to the tentative conclusion that both ethanol and adaptation, while having similar effects upon conditioning, do so either through separate mechanisms

(probably cortical) or differentially, through the same mechanism. If the first case were shown to be true, further doubt is thrown upon the adequacy of Spence's theory to explain the adaptation process. If the converse applied, however, this could be regarded as support for Spence's contentions. Eysenck's theory could probably accommodate either view.

Lack of ability to explain concisely where and how adaptation takes place does not detract from the probable significance of its action as a potential source of systematic error in conditioning experiments. If, for instance, nothing is known about the adaptive potential of the noxious UCS in use, the results of such experiments in which the number of CRs is correlated with, say a personality variable, are likely to attribute to that variable characteristics it does not possess. For example, in the Spencian tradition, a high anxiety S predictably forms more CRs than a low anxiety S. In the course of regular conditioning trials, however, adaptation must--according to the results of this study--operate as an uncontrolled variable. It is even probable that it operates differentially in the case of high and low anxiety groups. The same observation would, of course, apply equally to the results obtained from conditioning experiments in which Eysenck, Franks, et al, use Ss judged to fall at the extremes of the introversion-extraversion dimension.

If this interpretation of the effects of adaptation were found to be valid, it would seem that learning theories in general could benefit from a closer look at the nature of response decrement to

the UCS, which undoubtedly occurs in most classical conditioning situations.

In regard to the extinction data, these are no exception to the general observation that experimental extinction of the conditioned eyeblink response has typically been found to be rapid. As Spence (1963) has noted, in the majority of conditioning experiments, when extinction curves are plotted in terms of the number of previously non-reinforced trials, performance reaches its minimum level in as few as 6 trials, with many Ss ceasing to respond after the first non-reinforced trial. The latter was generally the case in the present study, thus extinction was too rapid to permit observation of the possible effects of the independent variables.

In attempts to explain rapid extinction, experiments by McAllister (1953), Reynolds (1958), and Spence (1963), have led to the conclusion that the underlying factor is a cognitive one. The S, apparently, is able to recognize a change in the experimental conditions with a shift from acquisition to extinction.

> Even when not aware of the fact their eyelids have been conditioned to respond to the CS, most subjects appear to become aware of it when, with the onset of extinction, the UCS is discontinued. (p. 1224, Science, 1963)

While this problem is worthy of further investigation for its own sake, a growing body of empirical data points to the inadequacy of extinction measures of the conditioned eyeblink response in human Ss as a means to understanding the learning process.

Finally, since the action of ethanol, regardless of dosage, has demonstrably been shown in this study to interfere with the learning of new response patterns, the possible relevance for such a finding for the field of alcoholism treatment cannot be overlooked. While it would not be argued (nor could it be) that the classical conditioning paradigm is adequate in itself to account for all learning, it would seem probable that the active alcoholic is severely limited in his capacity to learn from new experiences. This may well account for the typically regressive behaviour attributed to the alcoholic. If this is considered along with the possible effects of ethanol upon response patterns already established (an invitation to further research), it would seem that the alcoholic is undoubtedly suffering from the consequences of both processes (response prevention and response attenuation) when he presents himself for treatment. This, if a valid observation, indicates research into the efficacy of a directive treatment technique which would emphasize the learning process involved in the acquisition of simple new S-R bonds.

Conclusions

The results of this study show that the effects of experimentally controlled adaptation levels upon eyeblink conditioning performance are closely related to the action of ethanol, a central nervous system drug, upon the learning process.

The effect of adaptation trials upon acquisition of the conditioned eyeblink response--under the circumstances which this experiment was conducted--is as predicted by both Eysenck and Spence. Similarly, the effect of ethanol upon the same process is as predicted by both Eysenck and Spence. The results of this study, therefore, do not contradict either theory.

It is finally noted that the effects of adaptation and of ethanol, according to the findings of this investigation, are independent and additive.

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APPENDIX A1a

Blood alcohol levels of Ss in 0.05 alcohol group

No adaptation trials, blood alcohol levels 2, 3 & 4 taken 40, 60 & 100 minutes respectively after drinking began.

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.030	.055	.050	.052
2	.040	.060	.050	.055
3	.035	.050	.040	.045
4	.045	.050	.045	.047
5	.035	.052	.045	.048
6	.030	.049	.045	.047
7	.040	.050	.050	.050
8	.035	.050	.050	.050
9	.045	.052	.048	.050
10	.040	.050	.050	.050
11	.045	.045	.045	.045
12	.050	.050	.045	.047
13	.050	.060	.040	.050
14	.040	.045	.050	.047
15	.035	.045	.045	.045

35 adaptation trials, blood alcohol levels 2, 3 & 4 taken 40, 60 & 107 minutes respectively after drinking began

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.040	.052	.045	.048
2	.040	.060	.050	.055
3	.040	.055	.045	.050
4	.038	.055	.048	.052
5	.045	.060	.040	.050
6	.050	.060	.050	.055
7	.040	.055	.040	.047
8	.050	.045	.045	.045
9	.030	.050	.050	.050
10	.035	.048	.050	.049
11	.030	.050	.035	.042
12	.049	.044	.040	.042
13	.050	.055	.055	.052
14	.040	.050	.040	.045
15	.035	.050	.045	.048

APPENDIX A1b

Blood alcohol levels of Ss in 0.05 alcohol group
(continued)

70 adaptation trials, blood
alcohol levels 2, 3 & 4 taken
40, 60 and 114 minutes respectively
after drinking began

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.035	.060	.050	.055
2	.035	.060	.050	.055
3	.040	.055	.050	.052
4	.030	.050	.045	.048
5	.030	.050	.045	.048
6	.040	.045	.050	.048
7	.045	.058	.042	.050
8	.035	.050	.045	.047
9	.035	.045	.051	.048
10	.040	.065	.043	.054
11	.030	.045	.040	.042
12	.035	.050	.050	.050
13	.040	.052	.045	.048
14	.035	.055	.044	.049
15	.050	.060	.048	.054

APPENDIX A2a

Blood Alcohol levels of Ss in 0.10 alcohol group

No adaptation trials, blood alcohol levels 2, 3 & 4 taken 40, 60 & 100 minutes respectively after drinking began

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.090	.098	.080	.089
2	.090	.120	.100	.115
3	.070	.120	.100	.115
4	.100	.120	.120	.120
5	.070	.100	.095	.097
6	.065	.110	.100	.105
7	.070	.110	.090	.100
8	.070	.095	.110	.102
9	.065	.100	.095	.097
10	.075	.110	.090	.100
11	.070	.090	.090	.090
12	.080	.100	.090	.095
13	.070	.100	.085	.092
14	.080	.100	.097	.098
15	.075	.110	.100	.100

35 adaptation trials, blood alcohol levels 2, 3 & 4 taken 40, 60 & 107 minutes respectively after drinking began.

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.070	.095	.110	.102
2	.070	.110	.100	.105
3	.050	.090	.090	.090
4	.080	.100	.120	.110
5	.080	.110	.100	.105
6	.075	.100	.090	.095
7	.110	.130	.090	.110
8	.080	.110	.110	.110
9	.070	.110	.100	.105
10	.085	.100	.090	.095
11	.070	.110	.100	.105
12	.070	.095	.120	.107
13	.095	.110	.090	.100
14	.080	.110	.110	.110
15	.070	.110	.090	.100

APPENDIX A2b

Blood alcohol levels of Ss in 0.10 alcohol group (continued)

70 adaptation trials, blood alcohol levels 2, 3 & 4 taken 40, 60 and 114 minutes respectively after drinking began.

Ss	Blood Alcohol Levels			
	2	3	4	Av. of 3 & 4
1	.070	.100	.100	.100
2	.070	.120	.110	.115
3	.078	.100	.090	.095
4	.075	.100	.100	.100
5	.070	.100	.080	.090
6	.120	.100	.090	.095
7	.090	.110	.110	.110
8	.070	.110	.090	.105
9	.070	.100	.100	.100
10	.080	.100	.090	.095
11	.085	.110	.090	.100
12	.080	.095	.090	.093
13	.080	.120	.110	.115
14	.085	.120	.110	.115
15	.080	.100	.090	.095

APPENDIX B1

Conditioned responses given by individual Ss
in nine treatment groups

Group 1. No Alcohol

Ss	Adaptation Trials		
	Zero	35	70
1	84	60	27
2	89	82	57
3	85	30	62
4	25	65	73
5	70	71	93
6	87	48	64
7	87	30	60
8	95	66	25
9	95	81	84
10	94	93	32
11	80	49	85
12	84	81	41
13	59	92	65
14	95	93	38
15	80	82	64

Group 2. Blood Alcohol
level 0.05.

Ss	Adaptation Trials		
	Zero	35	70
1	10	60	14
2	50	69	82
3	35	30	83
4	75	49	43
5	33	66	9
6	71	79	74
7	25	54	70
8	71	48	51
9	68	69	16
10	82	32	53
11	81	29	27
12	83	83	13
13	93	32	33
14	33	72	79
15	40	47	33

APPENDIX B2

Conditioned responses given by individual Ss
in nine treatment groups

Group 3. Blood alcohol
level 0.10

Ss	Adaptation Trials		
	Zero	35	70
1	57	61	67
2	16	65	11
3	79	75	67
4	50	24	80
5	30	47	7
6	66	52	59
7	77	2	56
8	87	32	30
9	87	16	32
10	25	4	34
11	22	33	26
12	50	5	8
13	8	60	8
14	53	29	4
15	16	65	6

APPENDIX C

This questionnaire is designed to give us some idea of your normal drinking habits. Please be as accurate as you can in your estimates. Information will be treated as confidential.

1. How many years have you been drinking .....

2. Do you drink more or less than:

   a) one case of beer per week .....

   b) 13 Ozs. of spirits a week .....

   If you answer "more" to either of the above, please elaborate:

3. As described in B - would you consider that this habit has been fairly stable over the years .....

   If not - please elaborate:

4. Do you consider yourself to have the same (or similar) drinking habits as those normally around you?

   If not - please elaborate:

5. Do you consider yourself to be an average drinker .....

   If not, please explain.

6. On the whole, which of the following describes you best:

   a) very experienced drinker

   b) moderately experienced drinker

   c) a drinker of limited experience

Thank you.

APPENDIX D

Instructions to subject.

This is a study designed to investigate the effects of ethanol upon certain behaviour. At this time I am unable to tell you more about this--but will answer any questions you may have at the end of the experiment. Please be assured that nothing harmful or frightening will take place.

For ease of explanation, we may regard the experiment to consist of three phases. In phase 1, you are required to drink a glass of water, the purpose of which is to flush out the stomach. During the 30 minutes needed for this to take effect, you will be weighed and introduced to the Breathalizer, a machine used to calculate blood alcohol concentrations during the experiment.

In phase 2, you will be asked to consume the treatment beverage within a period of 20 minutes from the time drinking begins. During the 60 minutes which must elapse before phase 3, you will be encouraged to relax, using the facilities provided as you wish--radio, taped music, playing cards, 16 mm films.

Phase 3 will be explained at the time.

Phase 3 - the S is introduced in a general way to the conditioning equipment, seated, and attached to it. The instructions then follow:

Please listen carefully to the following instructions and follow them to the best of your ability. I cannot tell you the purpose of the experiment at this time, but again will gladly answer your questions at the end of the experiment.

APPENDIX D (continued)

Remain seated as quietly as possible and keep looking at the square object on the wall in front of you. Do not touch anything on your head. You will receive a series of stimuli during the experiment consisting of a tone and a puff of air. It is extremely vital that you do not try to control any of your natural reactions to these stimuli. Remain as relaxed as you can and let your reactions take care of themselves.

At any time you may communicate with me by speaking clearly into the microphone on the wall beside you. Would you now tell me what you are to do.

APPENDIX E

Name.

Phone.

Age.

Faculty

This is to certify that I am over the age of 21 years, that I am not an abstainer from alcoholic beverages, that, as far as I am aware, I am medically fit and free from any form of drug addiction. I voluntarily agree to participate in this undertaking in the full knowledge that I will be both paid (at the rate of $1.25 an hour), and expected to consume limited quantities of alcohol. I understand and further appreciate that if necessary I shall be detained by the experimenter, and at his discretion, to recover from the effects of alcohol.

Signed.

Date.

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